Research letters

Mental health of detained asylum seekers

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Asylum seekers arriving in the USA are likely to be held in detention for months or years pending adjudication of their asylum claims. We interviewed 70 asylum seekers detained in New York, New Jersey, and Pennsylvania. We used self-report questionnaires to assess symptoms of anxiety, depression, and post-traumatic stress disorder. At baseline, 54 (77%) participants had clinically significant symptoms of anxiety, 60 (86%) of depression, and 35 (50%) of post-traumatic stress disorder; all symptoms were significantly correlated with length of detention (p=0·004, 0·017, and 0·019, respectively). At follow-up, participants who had been released had marked reductions in all psychological symptoms, but those still detained were more distressed than at baseline. Our findings suggest detention of asylum seekers exacerbates psychological symptoms.

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Worldwide, there is a growing trend toward detention of asylum seekers arriving in industrialised countries for months or even years pending adjudication of their asylum claims.[1] In the USA, 5000 asylum seekers are estimated to be held in detention,[1–3] although reliable statistics on the number of detained asylum seekers are unavailable. Detention of asylum seekers has concerned health professionals and human rights advocates, in part because of the potential detrimental effects on the mental health of detainees.[1,3] Research on this subject, however, has been limited by difficulties in gaining access to detention centres. The Bellevue New York University (NYU) Program for Survivors of Torture and Physicians for Human Rights have done a systematic and longitudinal study of the effects of postmigration detention on the mental health of asylum seekers.

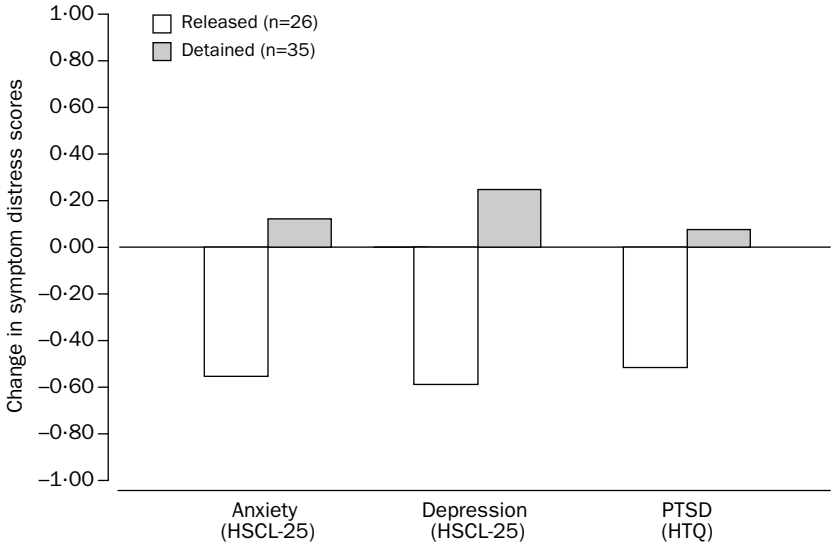
The US Immigration and Naturalization Services (INS) permitted access to detention facilities in New York, New Jersey, and Pennsylvania. These facilities included two INS detention centres run by private contractors. These jails are virtually windowless converted warehouses where only non-criminal INS detainees are incarcerated. Additionally, access was permitted to three local government-run jails in which criminals are also held. In all of these facilities, asylum seekers are heavily guarded, required to wear jail uniforms, and are shackled whenever they are transported outside of the detention facilities. The INS did not allow us access to a random sample of detained asylum seekers at these facilities. Therefore, we asked six local organisations providing pro-bono legal representation to detained asylum seekers to contact clients and ask about their willingness to be interviewed. Detainees were informed by researchers of the voluntary nature of the study and that participation would not affect their asylum applications. We obtained written informed consent from all participants. The study was approved by the Institutional Review Board of New York University School of Medicine and a review committee for Physicians for Human Rights.

Detainees were interviewed by physicians experienced in caring for refugees; translators assisted if necessary. Standardised psychological symptom measures were used: the Hopkins symptom checklist-25 (HSCL-25)[4] and the post-traumatic stress disorder subscale of the Harvard trauma questionnaire (HTQ).[5] Both measures have been used in studies of refugee populations and previously translated and back-translated in several languages, including French, Spanish, and Arabic. For participants who spoke other languages, scales were translated by the interpreter. Demographic information and history of

	Detained (n=35)		Released (n=26)	
	Number (%) above recommended cut-off*	Symptom scores, mean (SD)	Number (%) above recommended cut-off*	Symptom scores, mean (SD)
Baseline				
Anxiety	28 (80%)	2·40 (0·71)	19 (73%)	2·33 (0·72)
Depression	30 (86%)	2·52 (0·69)	22 (85%)	2·45 (0·65)
PTSD	19 (54%)	2·52 (0·62)	10 (39%)	2·45 (0·62)
Follow-up				
Anxiety	30 (86%)	2·58 (0·80)	9 (35%)	1·59 (0·56)
Depression	31 (89%)	2·73 (0·70)	10 (39%)	1·65 (0·59)
PTSD	21 (60%)	2·63 (0·71)	3 (12%)	1·80 (0·56)
Change in symptom scores†				
Anxiety	..	0·17 (0·61)	..	–0·75 (0·84)
Depression	..	0·21 (0·42)	..	–0·80 (0·81)
PTSD	..	0·12 (0·51)	..	–0·64 (0·64)

PTSD=post-traumatic stress disorder. *Cut-offs: 1·75 for HSCL-25 depression and anxiety subscales, 2·5 for HTQ. †p not significant for changes in any symptom score in detained group. p=0·0001 for all three symptom score changes in released group. Mean (SD) values are group mean at each assessment. Change=change in mean score from baseline assessment.

Psychological symptoms of asylum seekers still detained and those released at follow-up



Changes in psychological distress at follow-up
PTSD=post-traumatic stress disorder.

traumatic experiences were elicited from participants' asylum applications. Detainees who could be located were followed up 2 months or more after the initial interview to assess psychological changes.

Spearman correlation coefficients were used to analyse the relation between length of detention, which had a skewed distribution, and psychological distress. Independent sample *t* tests were used to assess differences in psychological distress between baseline and follow-up for patients who had been released and those still detained at follow-up.

Between Jan 1, 2001, and June 15, 2002, 87 detainees (73% of the caseload for the six agencies providing referrals) were referred to the study. Of these 87, 17 were excluded from the study: one was deported; ten were released before interview; one had been granted asylum but was still awaiting release; three did not complete the interview questionnaires; one withdrew the asylum claim; and one lost pro-bono legal support. Analyses are based on the remaining 70 participants (56 male, 14 female, mean age 28 years [SD 7·3; range 15–52]). Follow-up interviews were done at a median of 101 days (62–299) with 61 participants; 35 were still in detention and 26 had been released. Of the 26 released, 22 had been granted asylum and four released without asylum for medical or humanitarian reasons. Of the nine participants lost to follow-up: two had been deported; five could not be located; one had been transferred to another facility; and one refused to be interviewed. In April, 2003, 40 (57%) of the 70 participants had been granted asylum in the USA, and 14 individuals (20%) had been denied asylum and deported. Of the 40 individuals granted asylum, the median length of detention had been 7 months (2–42).

61 (87%) participants were detained in two INS detention centres and nine (13%) in three local government-run jails. The median length of detention before initial interview was 5 months (1–54). Most participants were from Africa (n=54), seven were from eastern Europe, four from Asia, two from the Middle East, and three from South America. 28 interviews were done in English, 17 in French, seven in Arabic, and 18 in other languages.

52 (74%) detainees had been tortured before immigration, 47 (67%) had been imprisoned in their native country, 41 (59%) reported the murder of a family member or friend, and 18 (26%) reported sexual assault. 54 (77%) detainees had clinically significant symptoms of anxiety, 60 (86%) of depression, and 35 (50%) of post-traumatic stress disorder. 18 (26%) participants reported thoughts of suicide while in detention, and two reported having attempted suicide.

49 (70%) participants perceived their mental health as having worsened substantially while in detention, and this perception was supported by Spearman correlations between length of time in detention and baseline levels of anxiety (r_s=0·34, p=0·004), depression (0·28, p=0·017), and post-traumatic stress disorder (0·28, p=0·019). Baseline mental health scores did not differ significantly between detainees eventually released and those who remained in detention, although differences were significant at follow-up (table). Participants still detained at follow-up had increased symptom scores for anxiety, depression, and post-traumatic stress disorder, whereas those who had been released had lower scores on all three scales (p<0·0001; figure).

Nearly all the detainees in our study had clinically significant symptoms of anxiety, depression, or post-traumatic stress disorder, which worsened with time in detention and improved on release. Our findings support anecdotal observations of other researchers and highlight the concerns raised by health professionals about the adverse effect of detention on asylum seekers.[1,2]

A limitation of our study is that there was no comparison group of non-detained asylum seekers. Although our sample of released participants was confounded by the fact that most were also granted asylum, the significant correlation between symptom severity at baseline and length of time in detention supports the hypothesis that detention significantly contributed to psychological distress. However, our reliance on self-report questionnaires rather than diagnostic interviews might have increased the proportion of individuals assessed as having clinically significant distress. Additionally, sampling was not random, which might limit the general applicability of our results to the entire population of detained asylum seekers. Furthermore, although we have no reason to think that detained asylum seekers represented by pro-bono legal groups differ from other detained asylum seekers, we cannot be certain.

Some participants could have deliberately exaggerated psychological symptoms, past traumatic experiences, or both, in order to bolster their asylum claims—despite being informed that their asylum application would not be affected by their responses. Nevertheless, the large proportion of participants ultimately granted asylum lends credence to their reports. Furthermore, there was no difference in reported distress at baseline or premigration traumatic experiences between detainees who were or were not granted asylum. Finally, despite a marked reduction in symptoms after release, many participants still had high levels of psychological distress, suggesting that symptom endorsement was not solely motivated by secondary gain.

Despite the limitations of this study, our results suggest that detaining asylum seekers exacerbates symptoms of depression, anxiety, and post-traumatic stress disorder in this vulnerable population. Our findings suggest that

policies concerning detention of asylum seekers should be reviewed, and highlight the need for mental health intervention to address the psychological needs of these individuals.

Contributors

A Keller, D Ford, C Trinh-Shevrin, J Leviss, H Smith, K Allden, and G Kim conceived and designed the study. A Keller, C Meserve, E Sachs, J Leviss, E Singer, H Smith, and J Wilkinson participated in the acquisition of data. E Sachs coordinated data collection. C Trinh-Shevrin and B Rosenfeld did statistical analyses. A Keller, B Rosenfeld, C Trinh-Shevrin, D Ford, C Meserve, J Leviss, E Sachs, and B Rosenfeld participated in analysis and interpretation of data. All authors helped to write the report.

Conflict of interest statement

None declared.

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Association of intercellular adhesion molecule-1 gene with type 1 diabetes

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Intercellular adhesion molecule-1 (ICAM-1) functions via its ligands, the leucocyte integrins, in adhesion of immune cells to endothelial cells and in T cell activation. The third immunoglobulin-like extracellular domain binds integrin Mac-1 and contains a common non-conservative aminoacid polymorphism, G241R. Phenotypically, ICAM-1 has been associated with type 1 diabetes, a T-cell-mediated autoimmune disease. We assessed two independent datasets, and noted that R241 was associated with lower risk of type 1 diabetes than is G241 (3695 families, relative risk 0·91, p=0·03; 446 families, 0·60, p=0·006). Our data indicate an aetiological role for ICAM-1 in type 1 diabetes, which needs to be confirmed in future genetic and functional experiments.

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The molecular mechanisms underlying type 1 diabetes are partly known. Three disease loci have been identified so far: the human leucocyte antigen (HLA) complex, the variable number tandem repeat locus located in the promoter region of the insulin gene (*INS*), and the cytotoxic T lymphocyte-associated antigen-4 gene (*CTLA4*).[1] The known functions of these genes suggest that T-cell activity is an important pathway in development of type 1 diabetes. Results of research in the mouse shows that ICAM-1 function during immune priming is necessary for the generation of effector T cells capable of destroying pancreatic insulin-producing β cells.[2] Genetic analysis of the ICAM-1 gene in families affected by type 1 diabetes could indicate whether its function has a causal role in the disease.

ICAM1 is located on chromosome 19p13 in a region that has shown some evidence of linkage to type 1 diabetes.[1] Two non-synonymous single nucleotide polymorphisms are known to be frequent in European populations: G→A in exon 4 encoding G241R, and A→G in exon 6 encoding K469E (rs1799969 and rs5030382, respectively; http://www.ncbi.nlm.nih.gov/SNP/). The K469E polymorphism has been investigated in type 1 diabetes in small samples, with variable results.[3] We assessed association of these two *ICAM1* coding polymorphisms in a sample of 3695 families affected by type 1 diabetes.

All family members were white, from Europe or the USA, with at least one affected child in every family (table). Mean age at onset of the affected offspring was 9·3 years (range 0–50). We obtained approval from the relevant research ethics committees and written informed consent of participants. DNA samples were genotyped with Invader (Third Wave Technologies, Madison, WI, USA) and TaqMan (Perkin Elmer Applied Biosystems, Foster City, CA, USA) assays. We recorded 99·5% concordance between these methods in 1242 samples tested to assess error in genotyping of the G241R polymorphism. Statistical analysis was done with STATA (version 8.1). Calculations of p values and 95% CI of relative risk (RR) were based on robust variance estimates, used to correct for clustering of affected individuals within families.

K469E did not show any association with the disease: K469 was transmitted 1931 times of 3897 (49·6%, p=0·59). By contrast, R241 was transmitted significantly less often: 938 transmissions of 1974 (47·5%, p=0·03; table). To control for the possibility that genotyping error or transmission ratio